A review of the anomalous origin of the left coronary artery from the anterior sinus of valsalva: Is prevention possible ?

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The origin of the left coronary artery from the anterior sinus of Valsalva is a rare coronary anomaly. Nevertheless, it remains an important pathological entity because of the possibility of its clinical and surgical consequences. Physicians should therefore be aware of this condition and consider it in their differential diagnosis of ischemic heart disease. This is a case report and review of the literature.

Introduction

The purpose of this paper is to provide an overview and to familiarize physicians with this anomaly. A review of 65 autopsies and cases of sudden death among these patients is included. In addition, we report a case of anomalous origin of the left coronary artery from the anterior sinus of Valsalva in Honolulu, Hawaii.

Case Report

An unmarried, 38-year-old Filipino woman was admitted to the hospital with a long-standing history of angina at rest and upon exercise. She had a history of diabetes, hypertension, and anemia; her only current medication was Nitrostat. Her past medical history included a tubal ligation in 1975 and two bladder infections, the last one in March 1990. Her family history was significant for sudden death—her mother died at age 45 of heart disease and the patient's child died at the age of 1 of an unknown cause.

Her blood pressure was 110/70, pulse 60 and regular, respirations 20, and temperature 98.7°C. The physical examination was otherwise unremarkable. Laboratory findings included a CBC, serum chemistries, serum electrolytes, and urinalysis were within normal limits. Echocardiography revealed mild cardiomegaly with left ventricular enlargement. The patient also had a positive thallium stress-test.

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Submitted June 1992

Angiography was performed to rule out coronary artery disease or an equivalent. It revealed normal left ventricular function and a large right coronary artery. There was a small left coronary artery arising from the right sinus of Valsalva that passed between the great vessels and provided primarily a circumflex distribution. (Figure 1: x-ray).

Surgical correction consisted of a coronary artery bypass graft. The left internal mammary artery was anastomosed to the left anterior descending coronary artery and a saphenous vein segment was used to connect with the circumflex system. The patient's postoperative course was uneventful with the exception of a single episode of low serum glucose (46 mg/dl). She was discharged 6 days later on Ecotrin and Persantine.

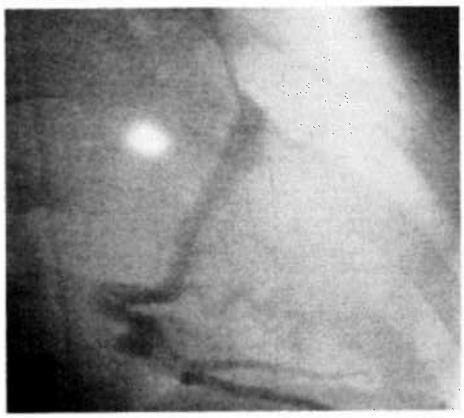

Figure 1: x-ray

Discussion

Normal coronary arteries have an architecture that is "...observed in at least 1% of unselected cases"[1]. This definition allows for *normal variants*; therefore, any variation that occurs in less than 1% is an anomaly[1]. The incidence of coronary anomalies has ranged from 0.28% to 1.2%[2-10]. Coronary anomalies can be divided into 3 categories:

1) Associated: A variation in response to a primary cardiac pathology.
2) Major: An abnormal coronary artery connection with a cardiac chamber or an abnormal origin from the pulmonary artery.
3) Minor: An abnormal origin from the aorta but with a normal distal circulation.

Anomalous origin of the left coronary artery from the anterior sinus of Valsalva fits into the third category. Its inci-

dence has been reported between 0.02% and 0.19%[2,3,4,9,11,12]. It is considered to be a minor anomaly because the blood entering both coronary arteries is fully oxygenated; this variation is compatible with life. However, numerous studies have documented that it is not such a benign condition after all. Cheitlin reported a 27% risk of sudden death in patients with this anomaly[13].

The various routes an anomalous left coronary artery can take will be discussed prior to addressing possible mechanisms of these sudden deaths.

There are 4 pathways an anomalous left coronary artery can take as it travels from its origin in the sinus of Valsalva to its final destination by dividing into the left anterior descending and circumflex arteries. The first is known as the retroaortic pathway (Figure 3). The left coronary artery leaves the anterior sinus of Valsalva and courses posteriorly around the aorta to its normal distribution. Presently, no cases of sudden death in patients with this pathway have been reported. In addition, Murphy reported a myocardial infarct over the distribution of the left coronary artery in one such patient. Symptoms were alleviated with bypass surgery of the anomalous left coronary artery although both coronary arteries were devoid of atherosclerosis[14]. This strongly suggests that the ischemia was caused by the retroaortic anomaly.

The second course an anomalous left coronary artery can take is called the anteropulmonic pathway (Figure 4). The left coronary artery leaves the sinus of Valsalva and travels anteriorly to the pulmonary artery to its normal distribution. Roberts reported sudden death in one such patient[15]. Pachinger presented a patient with such an anomalous left coronary artery who had angina despite patent coronary arteries[16]. Chaitman reported myocardial infarction in the left coronary artery's distribution in another patient with patent coronary arteries[2]. These studies of ischemia in the presence of patent but anomalous coronary arteries point to the possible cause of sudden death. Roberts and Kragel also reported sudden death in one such patient[15]. This evidence indicates that the anteropulmonic route of an anomalous left coronary artery is not a benign condition.

The third course is known as the interarterial pathway, in which the left coronary artery courses between the aorta and pulmonary artery (Figure 5). This is the most hazardous form and is responsible for a vast majority of the sudden deaths attributed to an anomalous left coronary artery from the anterior sinus of Valsalva.

There is a fourth, benign type of anomalous left coronary artery that has a septal pathway (Figures 6 and 7). The left coronary artery leaves the aorta and burrows into the ventricular septum, and it emerges anteriorly to branch into the left anterior descending and circumflex arteries which follow their usual courses. In a review of the literature, there has been only one

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Legend : All superior views

CX	-	Circumflex
LAD	-	Left Anterior Descending Artery
LMC	-	Left Main Coronary Artery
RCA	-	Right Coronary Artery
PA	-	Pulmonary Artery
LSV	-	Left Sinus of Valsalva
RSV	-	Right Sinus of Valsalva

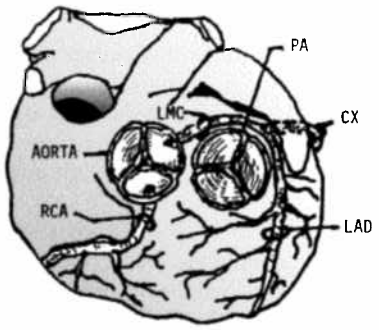

Figure 2: Normal anatomy of the coronary arteries.

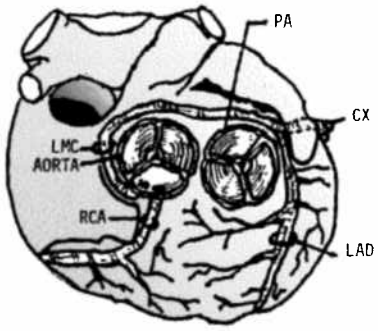

Figure 3: Anomalous left main coronary artery coursing posterior to the aorta.

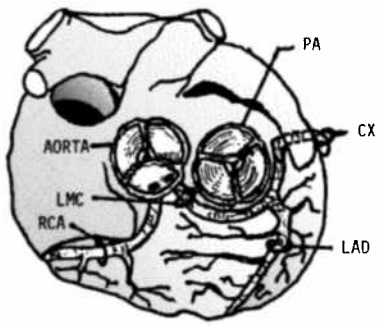

Figure 4: Anomalous left main coronary artery coursing anterior to the pulmonary artery.

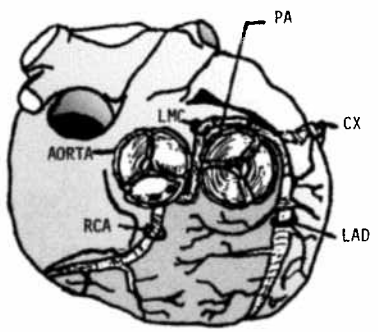

Figure 5: Anomalous left main coronary artery coursing between the aorta and pulmonary artery.

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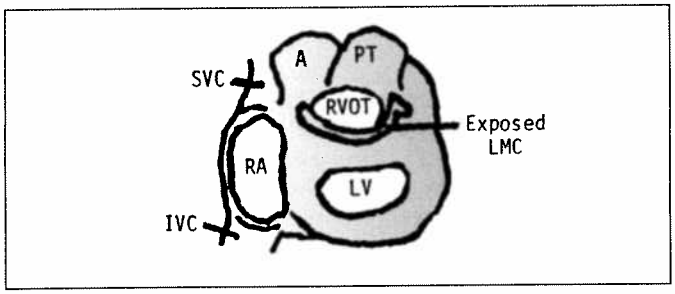

Figure 6: Transverse section through heart showing anomolous LMCA coursing in the ventricular septum view from below..

Figure 6 legend

A	—	Aorta
PT	—	Pulmonary
SVC	—	Superior Vena Cava
IVC	—	Inferior Vena Cava
LMC	—	Left Main Coronary Artery
CX	—	Circumflex Artery
LAD	—	Left Anterior Descending Artery
RVOT	—	Right Ventricular Outflow Tract
RA	—	Right Atrium
RV	—	Right Ventricle
LV	—	Left Ventricle

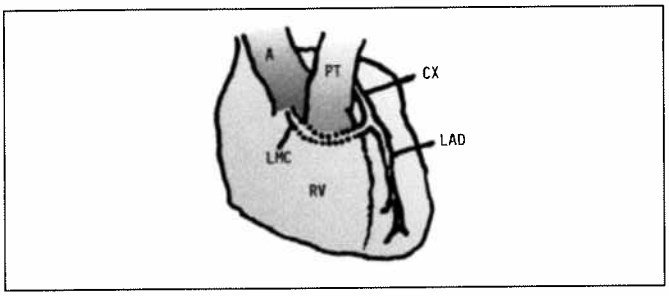

Figure 7: Anamolous left main artery coursing in the ventricular septum (anterior view).

patient documented with this septal pathway which might have contributed to his death[17]. This condition is considered to be relatively benign, and patients with the septal route typically live to advanced ages.

There has been much speculation as to the exact cause of sudden death in patients who have anomalous left coronary arteries originating from the anterior sinus of Valsalva. Most cases have been directly related to exercise. Of 39 cases of sudden death in patients under the age of 30 reviewed in the literature, 34 were exercise-related.

The most popular theory is that the anomalous coronary artery is compressed between the aorta and pulmonary artery during exercise[3,18,19,30]. Ischemia could be caused by the unsatisfied need for increased oxygen consumption by the myocardium and in peripheral vasodilation, or by an increase in systolic pressure leading to compression of the anomalous coronary artery.

Murphy[14] documented a case in which the density of contrast medium in the anomalous left coronary artery clearly diminished during systole.

This explains why sudden death almost never happens in patients with an anomalous septal left coronary; the buried left artery might be protected from compression.

Cheitlin disagreed with the compression theory and said that the pulmonary artery, a low-pressure system, would be an unlikely candidate in the compression of the left coronary artery[13]. However, patients with normal right ventricles and pulmonary vasculature have been found to develop marked transient pulmonary hypertension both before and after a coronary bypass operation[11]. Corday and associates have found that pulmonary hypertension can compress the coronary arteries[20]. Thus, transient pulmonary hypertension with a physiologic increase in systolic blood pressure during exercise could compress the anomalous left coronary and hinder its flow.

Several authors have discovered atherosclerotic stenosis in the portion of the anomalous left coronary artery that passes between the aorta and pulmonary artery. These plaques were postulated to be the result of chronic compression[7,19]. This is unlikely to be the sole cause of sudden death, but in conjunction with left coronary artery compression could compromise coronary blood flow.

Another theory based on ischemia deals with the acute angle

SURGICAL APPROACH

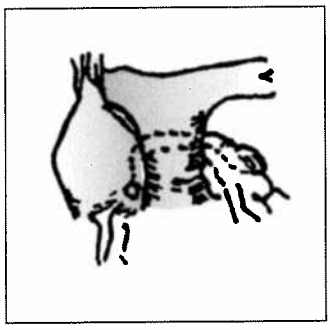

Figure 8a: Anamolous LMC

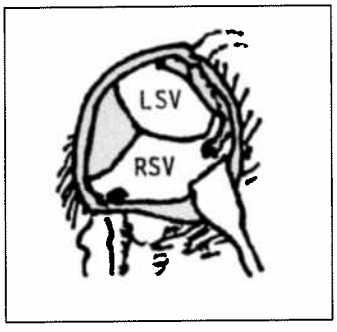

Figure 8b: Exposure of the anamolous LMC

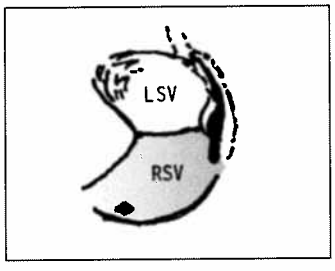

Figure 8c: The ostium is split and the incision is extended above the intercoronary commissure until the midpoint of the left coronary.

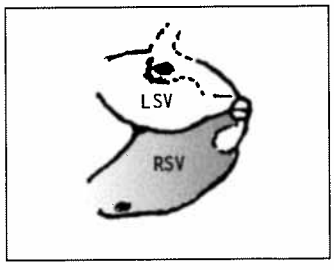

Figure 8d: The intima of the LMCA and aorta are joined, and the inter-coronary commissure is reattached

present in many cases as the artery leaves the aorta. An acute angle can be defined as an angle of less than 45 degrees between the aorta and the proximal portion of the left coronary artery[21]. Several authors have noted that an acute angle could lead to luminal narrowing and reduced myocardial blood supply from luminal kinking and intramural stretching as cardiac output and pressures are elevated during exercise[3,7,13,19,22]. Virmani, in a study of sudden death victims, found a significant increase in the incidence of such acute angles when compared with a control group[21]; acute angles were present in 18 of the cases reviewed[21]. A slit-like ostium was commonly found in association with an acute angle of the anomalous left coronary artery, often in conjunction with a flap-like closure. This condition was found in 11 of the cases reviewed, 10 of which possessed an acute angle. The already narrowed ostium could be narrowed further with exercise as the aorta distends, reducing coronary blood supply.

In addition, several authors have reported that the proximal left coronary artery that exits the ostium travels within the wall of the aorta, sharing a common intima[23,24]. With exercise, the aorta dilates, and as Sacks proposed, the intramural portion becomes flattened, reducing blood flow[23].

Virmani also described ostial ridges (defined as a ridge whose surface area occupied 50% of the coronary ostial area). In his study, 22 sudden death victims were compared with a control group of 19 patients who died of known causes. All 41 hearts were examined for abnormalities such as acute angle takeoff and ostial valve-like ridges. Patients with the anomalous left coronary artery with origin from the anterior sinus of Valsalva had a significantly higher incidence of ostial ridges than their control group counterparts. The ridges were thought to impede flow by compressing the left coronary artery as the aorta dilated during exercise[21].

Another possible cause for ischemia is congenitally hypoplastic coronary arteries. This is not the sole factor leading to sudden death in patients with anomalous left coronary arteries because only 5 of the cases possessed a left coronary artery significantly smaller than the

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right coronary artery. However, all 5 of these cases were associated with sudden death.

A final possible mechanism is coronary vasospasm. Maddoux and associates documented a single case of coronary vasospasm in an anomalous left coronary artery that led to myocardial ischemia[24].

It is important to keep in mind that all of these theories could be valid. Sudden death in patients with anomalous left coronary arteries cannot be explained by any one element alone; all of the aforementioned theories might play a role. The final pathway from ischemia to sudden death is commonly ventricular fibrillation[25].

It is essential to evaluate any patient with chest pain unattributable to typical causation because of the possible presence of a precarious anomalous left coronary artery. A workup needs to include a base ECG and a radionucleotide-scanning exercise stress test to locate regional perfusion deficits. In several cases, this has been helpful in revealing an anomalous left coronary artery[26]. However, several authors have reported myocardial infarction/sudden death in patients with anomalous left coronary arteries despite previous negative exercise stress tests[14,27,28].

Although anomalous left coronary arteries have been identified by echocardiography, angiography eventually will be necessary for a definitive diagnosis[7,26]. It is essential to define the exact course of the anomalous left coronary artery for both prognostic and surgical reasons. There are 3 components of direction in an anomalous vessel: Sagital, transverse, and coronal planes. Therefore, 2 views are necessary during angiography; the 2 most informative views are the right anterior oblique and the lateral projections[29].

The symptomatic young adult with an anomalous left coronary artery is of primary concern, whereas the same condition in adults is far less likely to result in sudden death. However, it is recommended that all patients undergo surgical correction. A thallium exercise-stress test should be performed. If positive, the anomalous vessel should be bypassed. However, if negative, the patient should be followed over time (Figure 8)[26].

The surgical alternatives are: Ostioplasty, separate coronary artery bypass grafting (CABG) to the left anterior descending and circumflex arteries, CABG to the left coronary artery and relocation of the ostium. Enlarging the ostium should be attempted only if the ischemia involved with an anomalous left coronary artery was caused by the slit-like ostium and the acute angle takeoff of the vessel. If the mechanism of ischemia is compression between the great vessels, such an operation would be ineffective. In a review of the literature, there were 3 documented cases of ostioplasty[13,32,33]. In each case, the patients improved postoperatively. In one of the cases, there was a 7-year follow-up on the patient which corroborated the success of the procedure.

Treatment with bypass grafting has received equal success. Sacks used a single graft to the left coronary artery and said this procedure would cause less restriction of flow than to apply separate grafts to the left anterior descending and circumflex arteries[23]. Nevertheless, there also have been many cases of separate bypass grafts that were extremely successful in alleviating symptoms of myocardial ischemia[7,11,24,26,29,33,34,35,36,37]. These results suggest that compression of the anomalous left coronary artery plays a significant role in myocardial ischemia.

Mustafa and colleagues suggest an innovative alternative[22]. The ostium of the left coronary artery is incised and separated from the anterior sinus of Valsalva and the incision extended to above the intercoronary commissure up to the midpoint of the left coronary sinus. At this point, the intima of the left coronary artery and the aorta are joined and the intrercoronary commissure reattached to the aorta (Figure 8). That patient did well postoperatively with a normal cardiac catheterization at 1- and 2-year follow-ups[22]. This procedure also was performed by Donaldson and associates with similar results[9].

Despite the success of these surgical techniques, it is difficult to judge which procedure is superior. There are no long-term follow-up results. The theoretical efficacy of one operation as compared to another would depend on which theory of the causation of ischemia in anomalous left coronary arteries was correct. The success of various surgical procedures supports the possibility that the ischemia could be caused by a combination of factors.

The ideal remedy would be primary prevention. This concept might appear to be far-fetched; however, as a result of significant advances in embryology and early detection of this anomaly, a preventive modality might loom. The first step toward achieving prevention must lie in understanding how the coronary arteries are formed and what circumstances can result in anomalies. If the causes of coronary anomalies can be discovered and techniques for predevelopmental detection developed, it is quite possible that someday such anomalies might be prevented.

Conclusion

Anomalous origin of the left coronary artery from the anterior sinus of Valsalva can be a serious entity that must be kept in mind in the differential diagnosis of any chest pain otherwise unexplainable. The anomaly is important in terms of the surgical implications, despite its rare occurrence. A high index of suspicion, a correct diagnosis and prompt surgical intervention could prevent sudden death. It is hoped when the embryology of coronary arteries and the causation of anomalies become understood in greater depth, a cost-effective means might be developed for the in-utero detection and prevention of these anomalies.

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